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Comparison of Risks from Outdoor and Indoor Exposure to Toxic Chemicals

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Environmental Protection Agency TEAM (Total Exposure Assessment Measurement) Studies have measured exposures of about 800 persons to 25 volatile organic compounds (VOCs) and exposures of about 300 persons to 32 pesticides. These persons were selected to represent more than 1 million residents of industrial manufacturing cities such as Bayonne and Elizabeth, New Jersey, and Los Angeles, California; cities with light industry, such as Greensboro, North Carolina, and Baltimore, Maryland; rural areas such as Devils Lake, North Dakota; and cities with high pesticide use such as Jacksonville, Florida, as well as low-to-moderate pesticide use such as Springfield, Massachusetts. The TEAM data provide an opportunity to estimate the risks from airborne exposure to a number of suspected carcinogens for a substantial number of persons residing in a wide variety of urban, suburban, and rural areas.

Because all of the TEAM Studies measured outdoor concentrations near the homes of the participants, it is possible to apportion the risks between outdoor and indoor sources. Upper-bound lifetime risks of cancer are calculated for both indoor and outdoor sources of 12 VOCs and about 23 pesticides measured in the TEAM Studies. These risk calculations are supplemented by calculations based on other studies for some additional pollutants, including radon and environmental tobacco smoke. The relationship of these upper-bound risk estimates to "best-guess" values is discussed. Sharper estimates of risk based on identifying population subgroups exposed to major sources are also discussed. Important gaps in our knowledge of exposure measurements are identified, e.g., particulates (including polyaromatic hydrocarbons); 1,3-butadiene, asbestos, chromium, cadmium, arsenic, vinyl chloride, methylene chloride, and most polar organics.

Introduction

For the last decade, the Environmental Protection Agency's (EPA) Total Exposure Assessment Measurement (TEAM) Studies have been providing data on the personal exposures (including indoor and outdoor concentrations) to organic chemicals for more than 1,000 persons representing more than 1,000,000 residents of 10 U.S. cities. About 35 of these chemicals cause cancer in animals and may cause cancer in man. In this paper, I calculate the upper-bound lifetime risk associated with airborne exposures to each chemical. I also try to apportion the risk between indoor and outdoor sources. Although the absolute magnitudes of these upper-bound risks are very uncertain, the relative rankings of the chemicals and their sources may be useful in focusing our attention on efficient ways to reduce exposure.

Methods

The calculation of cancer risk requires two factors: carcinogenic potencies of chemicals and mean exposures of people. Chemical potencies are taken from EPA sources (*1,2*). Exposure measurements (including some overnight indoor air measurements) for 12 volatile organic compounds (VOCS) are taken from TEAM Studies carried out in 8 U.S. cities between 1980 and 1987 (*3,4*). Personal exposures and indoor air concentrations for 23 carcinogenic pesticides were measured in two cities between 1986 and 1988 (*5*). Outdoor air measurements were made for all chemicals in the backyards of the subjects' homes; therefore, an estimate can be made of the relative contribution of outdoor and indoor air to total airborne exposure to all the target VOCs and pesticides.

In a previous study of cancer risks of six prevalent VOCs (*6*), the TEAM cities were divided into "metropolitan" and "non-metropolitan" categories. The mean exposures calculated for each city were averaged to provide a risk associated with each of the two categories. Assuming that the TEAM cities represented typical values, U.S. Census figures were employed to calculate a risk for the U.S. population. The results from that study have been reproduced here, with two additions: calculated risks from exposures to benzene during smoking and chloroform during showering. Both of these exposures could not be measured using the personal monitors employed in the TEAM Studies; however, they could be estimated using breath measurements for smokers (*7*) and models for exposure during showers (*8*).

An additional six VOCs have been added. Two of these (styrene and 1,1,1-trichloroethane) are prevalent, and their personal exposures and outdoor air concentrations are well characterized, but their carcinogenicity is in doubt. A third chemical (methylene chloride) is probably prevalent, but very few measurements of personal exposure have been made due to

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its high volatility. The remaining three chemicals (vinylidene chloride, 1,2-dichloroethane, and 1,2-dibromoethane) are well-established animal carcinogens but are much less prevalent. They have been measured in only a few percent of the personal and outdoor air samples collected in the TEAM Studies. Thus, the risk estimates for these six VOCs are more uncertain than the estimates for the original six VOCS.

The pesticide exposures are the unweighted means of the seasonally averaged values for each city. Since Jacksonville, Florida, was chosen as a high-use area and Springfield, Massachusetts, as a low-use area, the average of the two may represent a closer approach to actual mean exposures than either one separately.

Risks are calculated as a simple multiple of the exposure and the potency. If the potency is given in units of (milligrams/kilogram/day)$^{-1}$, the exposure is translated to a daily dose by assuming 20 m^3 inspired air per day and a body weight of 70 kg.

Results

The mean measured exposures, outdoor air concentrations, carcinogenic potencies, and calculated upper-bound cancer risks are displayed for 12 VOCs in Table 1 and for 23 pesticides in Table 2. Seven VOCs and four pesticides exceeded the *de minimus* or negligible risk level of 10^{-6} by a factor of 10 or more. The seven VOCs were benzene, vinylidene chloride, *p*-dichlorobenzene, chloroform, ethylene dibromide, methylene chloride, and carbon tetrachloride. The four pesticides were chlordane, heptachlor, aldrin, and dieldrin. All four pesticides have been banned by EPA. Despite the bans, exposures remain high, due perhaps to their long life in the soil and their movement into homes after being injected in the soil as termiticides. Carbon tetrachloride has also been banned from consumer products, but its long life in the atmosphere has led to a global background that is sufficiently high to result in a nonnegligible risk.

Three additional VOCs and four additional pesticides are at or above the 10^{-6} risk level, but by less than an order of magnitude. The three VOCs are tetrachloroethylene, trichloroethylene, and ethylene dichloride. The four pesticides (or degradation products) are dichlorvos, α-BHC, γ-BHC (lindane), and heptachlor epoxide. Two additional VOCs and four additional pesticides had upper-bound risks between 10^{-6} and 10^{-7}: styrene, 1,1,1-trichloroethane, hexachlorobenzene, propoxur, DDT, and DDE. Nine chemicals (all pesticides) were below the 10^{-7} risk level for airborne exposure.

Finally, one pesticide, pentachlorophenol (PCP), was never detected. However, the detection limit was very high for PCP, leaving open the possibility that this pesticide could represent nonnegligible lifetime risks of cancer. Additional exposure studies with lower detection limits for PCP, and also for some of the less prevalent VOCs such as ethylene dibromide and vinylidene chloride, are necessary before a more trustworthy estimate of their risks is possible.

Indoor sources accounted for the great majority (80–100%) of the total airborne risk associated with most of these chemicals. Carbon tetrachloride is the only one of the target chemicals for which outdoor sources account for a majority of the airborne risk, indicating the effectiveness of the ban on its use in consumer products.

Table 1. Upper-bound lifetime cancer risks of 12 volatile organic compounds measured in the TEAM Studies (1980–1987).

Chemical	Exposure, μg/mg³[a]	Potency, (μg/mg³)$^{-1}$ × 10^{-6}	Risk, × 10^{-6}	Outdoor air concentration[b]
Benzene				
Air	15	8	120[c]	6
Smokers	90	8	720[c]	—
Vinylidene chloride	6.5[d]	50	320	<1
Chloroform				
Air	3	23	70	0.6
Showers (inhalation)	2	23	50	—
Water	30[e]	2.3[e]	70	—
Food and beverages	30[e]	2.3[e]	70	—
p-Dichlorobenzene	22	4	90	0.6
1,2-Dibromoethane	0.05	510	25	0.03
Methylene chloride	6[f]	4	24	2[f]
Carbon tetrachloride	1	15	15	0.6
Tetrachloroethylene	15	0.6	9	3
Trichloroethylene	7	1.3	9	1
Styrene				
Air	1	0.3[g]	0.3	0.3
Smokers	6	0.3	2	—
1,2-Dichloroethane	0.5	7	4	0.2
1,1,1-Trichlorethane	30	0.003	0.1	7

[a]Arithmetic means based on 24-hr average exposures of ≈ 750 persons in 6 urban areas measured in the TEAM Studies.
[b]Based on backyard measurements in 175 homes in six urban areas.
[c]The risk estimates for benzene are based on human epidemiology and are therefore mean as opposed to upper-bound estimates.
[d]Six measurements exceeding 1000 μg/m³ were dropped from the calculation; inclusion of the measurements leads to an average exposure of 150 μg/m³.
[e]These figures are in micrograms per liter or parts per billion rather than micrograms per cubic meter.
[f]Based on only eight 24-hr measurements in 1987.
[g]Source: U.S. EPA (*21*).

Table 2. Upper-bound lifetime cancer risks from airborne exposures to 23 pesticides measured in the Nonoccupational Pesticide Exposure TEAM Study.

Pesticide	Exposure, ng/m³[a]	Potency, kg-day/mg	Risk, × 10^{-6}	Outdoor air concentration, ng/m³[b]
Banned termiticides				
Heptachlor	71	4.5	90 (19)[c]	7
Chlordane	198	1.3	70 (15)	14
Aldrin	13	17	60 (13)	0.1
Dieldrin	3	16	14 (3)	0.2
Heptachlor epoxide	0.4	9.1	1(0.2)	0.1
DDE	2.2	0.34	0.2(0.4)	ND[d]
DDT	0.7	0.34	0.1(0.02)	ND
Other pesticides				
Dichlorvos	33	0.29	2.7	ND
γ-BHC (lindane)	6.6	1.3	2.5	0.4
α-BHC	0.5	6.3	1	ND
Propoxur	100	0.0079	0.2	2.5
Hexachlorbenzene	0.3	1.67	0.1	0.1
Dicofol	2.6	0.34	0.05	ND
o-Phenylphenol	58	0.0016	0.02	0.6
2,4-D	0.6	0.019	0.003	0.1
Atrazine	0.05	0.22	0.003	ND
cis-Permethrin	0.4	0.022	0.003	ND
trans-Permethrin	0.1	0.022	0.001	ND
Chlorothalonil	0.7	0.011	0.002	0.5
Folpet	0.5	0.0035	0.0005	0.2
Captan	0.1	0.0023	0.00007	ND
DDD	<4	0.34	<0.4	ND
Pentachlorophenol	<730	0.13	<3	ND

[a]Arithmetic mean of population-weighted and seasonally weighted average personal exposures measured for 173 persons in Jacksonville, Florida, and 85 persons in Springfield/Chicopee, Massachusetts.
[b]Based on outdoor measurements at each home in the two cities.
[c]All risks calculated assuming 70-year lifetime exposure at the measured levels. For banned pesticides, whose environmental concentrations should decrease over time, an alternative calculation of risk (in parentheses) assuming a 10-year half-life in soil is provided.
[d]ND, not detected.

Discussion

Upper-Bound versus "Best-Guess" Estimates of Potency

The potencies employed in the risk calculations above are, with one exception (benzene), "upper-bound" potencies calculated from animal experiments. This raises the question of what the "best-guess" potency might be. Unfortunately, the EPA has chosen not to calculate best estimates of potency, arguing that such estimates are inherently more unstable than the upper-bound estimates. Without arguing this point, it may still be instructive to calculate best estimates of potency. For example, are such best estimates lower by a factor of 3, 10, or 100? Could the best estimate sometimes be 0? To answer such questions, we may examine the approach developed by a group at Harvard University (*9*). In this approach, a median potency is calculated from the animal studies, together with an estimate of the natural logarithm of the geometric standard deviation (σ_x) of that animal potency. A median potency in humans is then calculated, based on an assumed uncertainty connected with extrapolation from animal to man ($\sigma_y = 1.5$), and an uncertainty (if necessary) associated with converting from oral dose to inhalation dose ($\sigma_t = 1.6$). Assuming log-normal distributions for all the sources of uncertainty, estimates of mean and upper-bound potencies can be calculated using standard relationships of log-normal distributions:

$$\text{Mean} = \text{Median} \times \exp(\sigma^2/2) \quad (1)$$

where $\sigma^2 = \sigma_x^2 + \sigma_y^2 + \sigma_t^2$.

When these calculations are carried out, using values for the median potencies and their uncertainties as calculated by the Harvard group, we find that 95% upper-bound potencies are typically about seven times the mean potencies. This finding is in general agreement with the results of a study (*10*) that compared upper-bound potency estimates from animal studies with observed potencies from human epidemiology of about 20 chemicals that are both animal and human carcinogens. That study found that, in general, the upper-bound estimates from the animal data were about an order of magnitude higher than the best estimates from the human data.

Therefore, the upper-bound risk estimates in Tables 1 and 2 (except for benzene) may be divided by a factor of 7 or 10 to provide best estimates of risk based on mean potencies and mean exposures. This results in benzene emerging as the single chemical with the highest risk of all 35 considered.

For some of these chemicals, it is also possible to argue that the best estimate of risk is 0. For example, if a chemical causes cancer in animals but not in man, its carcinogenic risk in humans is 0 by definition. Now suppose that a chemical is "more likely than not" to be a noncarcinogen—what is the best estimate of its carcinogenic risk? One way to answer this question is to say that there is a better than 50% probability that it is not a carcinogen, and therefore the best (median) estimate of risk is 0. Another approach might be to assign a probability that it is a carcinogen, calculate the risk on the basis of the animal studies, and then dilute that risk by multiplying by the assigned probability. Thus, if the animal studies are ambiguous, and we assign only a 10% probability that the chemical is a human carcinogen, the risk calculation would be multiplied by 0.10 to arrive at a best estimate of risk. In the case of the chemicals considered here, most are classified as "B2" (probable human carcinogens), but some are classified as "C" (possible human carcinogens). (Still others, such as tetrachloroethylene and *p*-dichlorobenzene, wobble back and forth between the two classifications.) If the probable carcinogens were assigned a likelihood factor greater than 50%, and the possible carcinogens were assigned a factor less than 50%, one of the above two approaches could be employed to further adjust the risks calculated in Tables 1 and 2.

Assigning Risk to Population Subgroups

The risk calculations above have generally been made on the basis of the entire population studied in the TEAM Studies or on extrapolating those results to the U.S. population. One exception has been the risks to smokers of benzene and styrene, which apply only to the 50 million active smokers in the United States. However, if it were possible to identify sources of exposure and characterize population subgroups on the basis of their exposure to those sources, it would be possible to refine our estimates of risk. In particular, we would find that risks are higher among the exposed subgroups and lower (perhaps 0) among the less exposed or unexposed subgroups.

As an example of the above points, we may consider the pesticide dichlorvos. This pesticide was found in about one-third of Jacksonville homes and only 2% of Springfield homes. Outdoor concentrations were negligible in both regions. If the personal exposures came mostly from use of a consumer product containing dichlorvos, it seems reasonable to calculate a risk based on exposures to the users only. Thus, the calculated risk to Jacksonville users would be 3 times the risk averaged over the entire population, and the calculated risk to Springfield users would be 50 times the risk averaged over the population. Since the calculated risk for the Jacksonville population was 5×10^{-6}, the risk to users would be 15×10^{-6}; the calculated risk for the Springfield population was 3×10^{-7}, leading to a risk to users of 15×10^{-6}—identical to the risk to Jacksonville users. This calculation has not changed the total population risk in either case; it has simply apportioned the risk across the populations. Thus we have gained a sharper definition of the risk. It is also interesting to note that the risks calculated in this way show that in both areas, upper-bound risks to users exceed the one-in-a-million level by a factor of 15; the previous calculation indicated that the upper-bound risks averaged across the population were very close to this dividing line. This approach does not appreciably change the risks calculated for the more prevalent VOCs and pesticides, but has the potential for order-of-magnitude increases in calculated risks for the less prevalent chemicals.

For example, applying this approach to other pesticides, we find that α-BHC, which was found in only 27% of Jacksonville homes and only 2% of Springfield homes, has a calculated average risk in these homes of 8×10^{-6} and 20×10^{-6}, respectively, compared to the value of only 1×10^{-6} averaged across the population. Other pesticides whose lifetime upper-bound risks in exposed homes approached or exceeded one in a million included 2,4-D, DDE, hexachlorobenzene, and dicofol.

Uncertainty of Estimates

Great uncertainty accompanies most risk estimates. The major uncertainties involved in potency calculations are well known:

the extrapolation from animals to man and from high dose to low dose. These uncertainties are such that a given chemical may not cause human cancer at all; the actual cancer risk may be exactly 0. Even if the risk is not 0, the estimates could easily be wrong by factors of 10, 100, or more, depending on the shape of the dose–response curve, the possible existence of a threshold due to DNA repair or other mechanisms, and many other factors.

Considerably less uncertainty is associated with some of the exposure estimates. The overall mean VOC exposure in eight cities was usually within a factor of 3 of the extremes for an individual city, whether the city was rural, suburban, urban, or heavily industrialized. The reason for this predictability appears to be the relative importance of consumer products, personal activities, and building materials to human exposure; such factors do not vary greatly across the country. Of course, personal activities can result in very high exposures for short periods, but we are concerned here with long-term exposures. Somewhat more variation was noted for pesticides, with differences of a factor of 10 in exposure noted for a number of pesticides in Jacksonville and Springfield.

For the nine prevalent VOCs, about 2000 measurements have been made of 12-hr average personal exposure and more than 500 have been made of outdoor concentrations. Thus, for these more prevalent VOCs and pesticides, relatively little error is associated with the estimates of the relative contribution of indoor and outdoor sources. The reason is that the same instrumentation was used to measure both indoor and outdoor air. Even if the instruments were biased, the relative proportions would remain nearly unchanged.

However, two other VOCs were measurable in only a small percentage of samples: vinylidene chloride (7%) and ethylene dibromide (2%). (Ethylene dichloride was measured in about 20% of the samples, but most measurements hovered close to the detection limit.) For these more rarely found chemicals, the indoor/outdoor ratios are less certain, as are the risk estimates.

Some chemicals were prevalent but do not have sufficient animal studies to establish their carcinogenicity. Among these are toluene and xylenes, not found to be carcinogenic in 2-year rat and mouse studies conducted by the National Toxicology Program (NTP), but found to be carcinogenic in natural-lifetime rat and mouse studies carried out in Italy (C. Maltoni, personal communication). Limonene (used in lemon-scented products and also as a food additive) was the VOC with the highest average concentration in people's homes; a recent 2-year NTP study found clear evidence of carcinogenicity in one sex–species combinations but no evidence in the other three sex–species combinations. Two pesticides that were prevalent at relatively high concentrations indoors were chlorpyrifos (Dursban) and diazinon. Health studies of these pesticides do not completely rule out their possible carcinogenicity. Because of the prevalence and high concentrations of these VOCs and pesticides, further health studies are indicated. Several of the chemicals with the highest associated risks will be discussed separately.

Benzene

Only one of these chemicals is considered a human carcinogen: benzene. Therefore, the risk estimate associated with benzene is on more solid ground than any of the others. Benzene is also the only one of these chemicals with human epidemiological studies showing a possible influence of environmental levels of exposure on cancer risk: two studies show that children of smokers die of leukemia at two or more times the rate of children of nonsmokers (*11,12*). The higher mortality rate is consistent with the measured elevated levels of benzene in the breath of smokers (suggesting exposure of the fetus in the womb of the pregnant smoker). Elevated levels of benzene in the air of homes have also been documented by the TEAM Study and by a study in West Germany (*13*); however, the increase (on the order of 50% in both studies) does not seem enough to explain the increase in the mortality rate unless children are more susceptible to benzene-induced leukemia at some point in the first 8 to 9 years of life.

Major sources of exposure to benzene appear to be active and passive smoking, driving and other personal activities associated with automobiles, use of attached garages for parking cars, storing gasoline and kerosene, and the use of certain consumer products (marking pens, paints, glues, rubber products). The major outdoor source is auto exhaust; emissions from stationary sources account for only a few percent of nationwide exposures.

Vinylidene Chloride

Vinylidene chloride is highly volatile and therefore "breaks through" the Tenax monitor after only a portion of the monitoring period. The concentration is calculated on the basis of the "breakthrough volume" rather than the actual sampling volume and, depending on the pattern of exposure during the monitoring period, may be either an over- or underestimate of the actual concentration. Also, because the sampling volume of 20 L is effectively reduced to the breakthrough volume of only a few liters, the sensitivity is reduced by the same factor. The limits of detection for vinylidene chloride ranged from 3 to 14 $\mu g/m^3$, about an order of magnitude worse than for most of the other target VOCs. Out of 1085 personal air samples collected from 355 New Jersey residents over three different seasons, only 77 (7%) had measurable concentrations of vinylidene chloride. (Another 107 [10%] showed trace concentrations.)

The population risk for such rarely detected chemicals can be calculated, but the interpretation of the risk presents difficulties. For example, the single highest measured exposure to vinylidene chloride was 120,000 $\mu g/m^3$, which was incurred by a cabinet maker. (The second highest value of 14,000 $\mu g/m^3$ was also measured for this same person in a different season.) Taken together, these two values accounted for more than 80% of the total calculated exposure (and therefore the risk) for the population. If we include these values in our calculation of risk, then vinylidene chloride exposures average 150 $\mu g/m^3$, and the upper-bound risk is 7.5×10^{-3}, greater than the risks from radon and passive smoking combined. If we drop these two values, the population exposure decreases to 28 $\mu g/m^3$, and the risk of 1.4×10^{-3} is still very large. However, four other exposures exceeded 1000 $\mu g/m^3$. If these values are also dropped from the risk calculation, the average exposure decreases to 6.5 $\mu g/m^3$, and the associated upper-bound risk decreases to 3.2×10^{-6}. Thus, the population average of 150 $\mu g/m^3$ for all 355 persons is actually composed of an average of 6.5 $\mu g/m^3$ for about 350 persons, and an average of about 30,000 $\mu g/m^3$ for about 5 persons. This corresponds to a difference in risk of the two groups of a factor of 5000.

Because of the tremendous effect of a few measurements, the calculated upper-bound risk for vinylidene chloride must be considered tentative. Only additional data on personal exposures (collected by methods with a sensitivity of 1 $\mu g/m^3$ or less) will provide the information necessary for an adequate risk assessment.

p-Dichlorobenzene

p-Dichlorobenzene has two major uses: as a moth repellent, it is a registered pesticide and as an air freshener, it is an additive (often unlabeled) to consumer products. From the TEAM Study results, it appears that about one-third of homes use *p*-dichlorobenzene. Therefore, the average risk to users is about three times the risk shown in Table 1. The risk to nonusers should be no more than that associated with average outdoor concentrations or about 1% of the risk to users.

Chloroform

Chloroform is unique among the target VOCs in having many routes of exposure: air, food, water, and beverages. Indoor and outdoor air levels and levels in drinking water have been documented in all the TEAM Studies. The pilot TEAM Study of 1980–1982 also documented chloroform levels of 15 to 56 ppb ($\mu g/L$) in milk, butter, cheese, and ice cream and 9 to 178 ppb in soft drinks (*14*). A recent Japanese study (Y. Sato, personal communication) of seven housewives indicated that they were exposed to chloroform through all routes, but that the diet provided more exposure (10.7 μg/day) than the air (2.0) and water routes (2.4 μg/day).

Methylene Chloride

Methylene chloride is too volatile to be collected on Tenax; therefore, few personal exposure measurements have been made. Several indoor and outdoor measurements were made in the 1987 TEAM Study in Los Angeles using evacuated canisters; these are the values on which the risk estimate has been based. However, the existing data are so sparse that the estimate must be considered very speculative.

Short-term exposures at the high part per million level from using paint strippers have been documented (*15*). Such an exposure for 1 day would equal the lifetime exposure to ambient concentrations of methylene chloride. Therefore, the population risk from this chemical might better be calculated from data on the number of people who use paint strippers and the amount of time they use them.

Ethylene Dibromide (1,2-Dibromoethane)

Ethylene dibromide is widely used as a fungicide, particularly on grain, and therefore the main risk is thought to be through food. However, the potency is so high that even the very low airborne exposures measured in the TEAM Study produce a nonnegligible risk. Only 15 of 621 personal air samples (2.4%) exceeded the quantifiable limit of 0.05 $\mu g/m^3$. Another 61 samples (12.2%) showed trace amounts. If we assume a value of 0 for the 545 nondetected samples, and the lowest possible value of 0.05 for the 61 trace samples, the average exposure is 0.014 $\mu g/m^3$. Assuming the maximum values for the trace samples (0.24 $\mu g/m^3$) and the nondetectable samples (0.05 $\mu g/m^3$) results in an average exposure of 0.087 $\mu g/m^3$. A value between these two extremes is 0.05 $\mu g/m^3$, resulting in a risk estimate of 25×10^{-6}. Only 3 of 282 outdoor air samples were measurable (and only 5 were at trace levels), and the range of average values using the same assumptions as above is 0.003 to 0.06 $\mu g/m^3$. The maximum personal exposure was only 0.97 $\mu g/m^3$, so that the risk calculations are not extensively skewed by a few samples as they were in the case of vinylidene chloride.

Chlordane and Heptachlor

Chlordane and heptachlor were recently withdrawn (April 1988) after wide use as termiticides (approximately 85% of the market). They were applied primarily as a liquid poured or injected into soil around building foundations. Therefore, their appearance in the Nonoccupational Pesticide Exposure Study (NOPES) as airborne vapors may indicate widespread intrusion of soil gas into the home through cracks or drains in the basement or ground floor.

Aldrin and Dieldrin

Aldrin and dieldrin were withdrawn from use in the United States in the early 1980s. They were used mainly as termiticides (about 10% of the market). Their appearance in the NOPES study is further indication of a long half-life in soil coupled with some mechanism allowing intrusion into the home.

Dichlorvos

Dichlorvos was widely used on pest strips before such a use was banned. Measurements during different seasons in the two cities ranged from 98 to 99% not detectable in Springfield, and from 65 to 89% not detectable in Jacksonville. As discussed above, although the risk averaged over the entire population is close to the 10^{-6} level of risk, when averaged over the smaller population of users, the risk climbs to about 15×10^{-6} in both cities.

Exposures through Other Routes

All of the chemicals discussed above (both VOCs and pesticides) were measured in drinking water, and found to present less than 1% of the risk due to airborne exposures with the single exception of chloroform. All of the chlorinated VOCs were also measured in food and beverages; again chloroform was the only VOC found in significant amounts in food.

Exposures through routes other than air and water have been documented for some of the pesticides. Many of the pesticides have been measured in food by the Food and Drug Administration for years; however, exposures in food account for only a small proportion of total exposure to the four pesticides of highest risk through airborne routes. Food exposures outweigh air exposures for some of the other pesticides (e.g., Captan).

House dust may provide an important reservoir for any or all of the pesticides and possibly also for the least volatile of the VOCs (*p*-dichlorobenzene, tetrachloroethylene). DDT was found in house dust in five of eight homes in the NOPES study. As this chemical has been banned for nearly two decades, its appearance in house dust is troubling. Ingestion of the dust by

toddlers could be an important additional source of risk. The DDT may be tracked in on people's shoes from outdoor soil.

Comparison with Other Environmental Risks

Several organic chemicals of interest were not monitored in the TEAM Studies. Among these are formaldehyde and 1,3-butadiene. Risk estimates for these chemicals may be compared to the risks calculated above.

The carcinogenicity of formaldehyde is controversial, due to the unusual metabolic pathway associated with its carcinogenicity in rodents. Estimates for the cancer risk of formaldehyde (*16*) range over extreme limits, from 0 to 10^{-3}. Employing an intermediate potency factor (unit risk of 1.3×10^{-5} $[\mu g/m^3]^{-1}$), measured values of 40 $\mu g/m^3$ for normal (non-mobile home) housing stock results in a risk of about 5×10^{-4}. (Average outdoor concentrations of formaldehyde have been about 4 $\mu g/m^3$, corresponding to a risk of about one-tenth of this level.)

Recent animal studies of 1,3-butadiene have resulted in revising its potency upward by nearly three orders of magnitude. Although no personal exposure data are available for this chemical, a recent study has measured the level in sidestream smoke at about 400 μg per cigarette (*17*). This is approximately the level of benzene in sidestream smoke (330 μg per cigarette); therefore, if 1,3-butadiene is not too reactive, we can calculate that it will be elevated by about 4 $\mu g/m^3$ in smoking homes and by 13 $\mu g/m^3$ in workplaces allowing smoking. Using the revised unit risk value of 2.8×10^{-4} and assuming 38 million homes with smokers averaging three residents each, 75 million workers in workplaces allowing smoking, and 26 million nonworkers exposed to cigarette smoke, we arrive at an upper-bound risk associated with exposure to 1,3-butadiene in environmental tobacco smoke of 6×10^{-4}. No data exist on personal exposures or indoor concentrations of 1,3-butadiene. (Outdoor concentrations of this chemical have been estimated to lie within a range of 0.3 to 1.6 $\mu g/m^3$, corresponding to a risk of about $1\text{–}4 \times 10^{-4}$.)

Thus, the individual risks for formaldehyde and 1,3-butadiene are greater than the airborne risk of any of the other 35 VOCs and pesticides considered in this report. However, the great uncertainty in the carcinogenic potency of formaldehyde, including the uncertainty as to whether it is a human carcinogen at all, and the lack of exposure data for 1,3-butadiene make the risk estimates for these two chemicals particularly speculative.

The combined upper-bound risk of about 10^{-3} associated with these 37 predominantly indoor organic chemicals appears to be similar to the risks associated with the most severe environmental hazards (radon and passive smoking). For example, the risk associated with nonsmokers' exposure to radon has been estimated to be about 10^{-3} and that with passive smoking has been estimated (*18*) at 2×10^{-3}. It should be noted, however, that the risk estimates for radon and passive smoking are based on human epidemiology studies, and are therefore on firmer ground than all of the risk estimates for the organic chemicals with the exception of benzene.

The risk estimates for these organic chemicals are considerably higher than the risks associated with some EPA regulations (National Emission Standards for Hazardous Air Pollutants [NESHAPS] and Superfund clean-up criteria). Similar conclusions regarding the importance of indoor air pollution compared to other environmental hazards have been reached by EPA headquarters (*19*) and by three EPA regions (*20*); both of these reports rank indoor air pollution as among the top two or three environmental threats to public health.

Two other risk estimates for personal exposure to VOCs have been published (*9,16*). McCann et al. (*16*) arrived at similar risk estimates for most of the chemicals; Tancrede (*9*) estimated 5 to 10 times higher risks, due partly to using a different method for calculating potencies from animal data and partly to considering explicitly several additional sources of uncertainty. No previous risk estimates for most of these pesticides have been possible due to the lack of exposure information.

Actions to avoid these risks may be taken by individuals. Since the sources of the risks are often personal activities (smoking, using air fresheners), these activities can be halted or modified. (For example, smokers could establish a room in the home with separate ventilation.) Exposures from chloroform could be reduced by drinking bottled water or using an activated carbon filter on the water supply. Exposures from petroleum-based products could be reduced by discarding or storing used paint cans and sprays in a detached garage or tool shed. Dry-cleaned clothes could be hung outdoors for a day (one study indicates that 20–30% of tetrachloroethylene residues on the clothes will outgas during the first day).

The reason for the large number of pesticides observed in indoor air in the latest TEAM Study is not well understood. Termiticides, like radon gas, may be entering the basement due to soil gas movement; it may be that the same techniques to control radon (sealing the foundation, providing separate ducting at the entrance points) may also control termiticide entry. Other pesticides, particularly the long-lived chlorinated hydrocarbons such as DDT, may be entering the home by being tracked in on people's shoes. If so, removing shoes before entering the home, and reducing or eliminating the use of carpets or rugs (which collect large amounts of dust containing pesticides and metals as well), should reduce pesticide exposures.

Summary and Conclusions

Measured personal exposures to 12 VOCs and 23 pesticides in EPA's TEAM Studies have been used to arrive at upper-bound lifetime cancer risk estimates. Seven VOCs and seven pesticides have upper-bound risks ranging from 10^{-6} to 10^{-4}. The combined upper-bound risk of about 10^{-3} from these organic indoor air pollutants is nearly comparable to the estimates of risk from radon and environmental tobacco smoke. (However, the latter two estimates are based on human epidemiology studies and are therefore subject to far less uncertainty.) These upper-bound risks are much greater than the health risks associated with most other environmental problems.

Several chemicals for which we have inadequate information, either on exposure or potency, to calculate risk were identified: vinylidene chloride, methylene chloride, 1,3-butadiene, formaldehyde, ethylene dibromide, chlorpyrifos, and diazinon. Despite the recognized large uncertainty in these risk estimates, these findings provide additional support for the conclusion of two recent comparative rankings of environmental risk by EPA: that indoor air pollution is one of the greatest threats to public health of all environmental problems.

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